CLINICAL STUDY ON DERMATOLOGICAL PRESENTATION IN 100 CASES OF HIV INFECTION

DISSERTATION

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CERTIFICATE

This is to certify that this dissertation entitled **"CLINICAL STUDY ON DERMATOLOGICAL PRESENTATION IN 100 CASES OF HIV INFECTION"** is a bonafide work done by **DR. LINCY C.F,** Post Graduate in M.D. Dermatology, Venereology and Leprosy, Coimbatore Medical College, Coimbatore- 641014, during the academic year 2009-2010. This work was done under my direct guidance and supervision and submitted for the M.D.BRANCH XII A examination in April 2011 to the Tamil Nadu Dr.M.G.R. Medical University, Chennai.

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DECLARATION

I **Dr.LINCY C.F,** solemnly declare that this dissertation titled **"CLINICAL STUDY ON DERMATOLOGICAL PRESENTATION IN 100 CASES OF HIV INFECTION"** is a bonafide work done by me at Coimbatore Medical College during 2009-2010 under the guidance and supervision of Prof. Dr.P.P.Ramasamy, M.D., D.D., Professor and Head, Department of Dermatology, Coimbatore Medical College, Coimbatore-641014.

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CONTENTS

Introduction

INTRODUCTION

Acquired immunodeficiency syndrome (AIDS) was recognized as a new disease for the first time in 1981.[1] AIDS was recognized because in March 1981 eight cases of more aggressive form of Kaposi's Sarcoma had occurred amongst young gay men in New York and at the same time there was an increase in the number of cases of Pneumocystis Carinii pneumonia (PCP) in both California and New York.[2, 3] Despite the development of laboratory methods, dermatological symptoms were in the past and still are the basic indicators of presence and physical course of the disease; human immunodeficiency virus (HIV) infection and AIDS are frequently related to a wide range of skin and mucosal manifestations.[1] This ranges from macular, roseola like rash in the acute seroconversion syndrome to the extensive end-stage Kaposi's Sarcoma.[4]

In primary HIV infection manifestations like fever, joint pain and night sweats are non specific for identifying acute seroconversion, because in developing countries where other endemic diseases with similar complaints are common.[5, 6] In the developed countries, primary HIV-1 infection is estimated to be symptomatic in up to 80% of cases [Acute retroviral syndrome (ARS)] [7] with the advent of Highly Active Anti Retroviral Therapy, the course of HIV/AIDS has been significantly changed.

In this study an attempt is made to find out the various dermatological manifestations associated with HIV/AIDS and to identify the most common predictor of HIV/AIDS in this part of the country.

Literature Review

LITERATURE REVIEW

Epidemiology of HIV/ADIS**:**

Ever since its recognition in 1981, HIV/ADIS Continues to ravage all the continents of the world.[8] More than 25 million people have died of AIDS since 1981.[9]

World Wide Statistics:

At the end of 2008, women accounted for 50% of all adults living with HIV worldwide. Africa has over 14 million AIDS orphans. The number of people living with HIV has risen from around 8 million in 1990 to 33 million today, and is still growing. Around 67% of people living with HIV/AIDS are in Sub Saharan Africa. In developing and transitional countries, 9.5 million people are in immediate need of life saving AIDS drugs; of these only 4 million (42%) are receiving the drugs.[9]

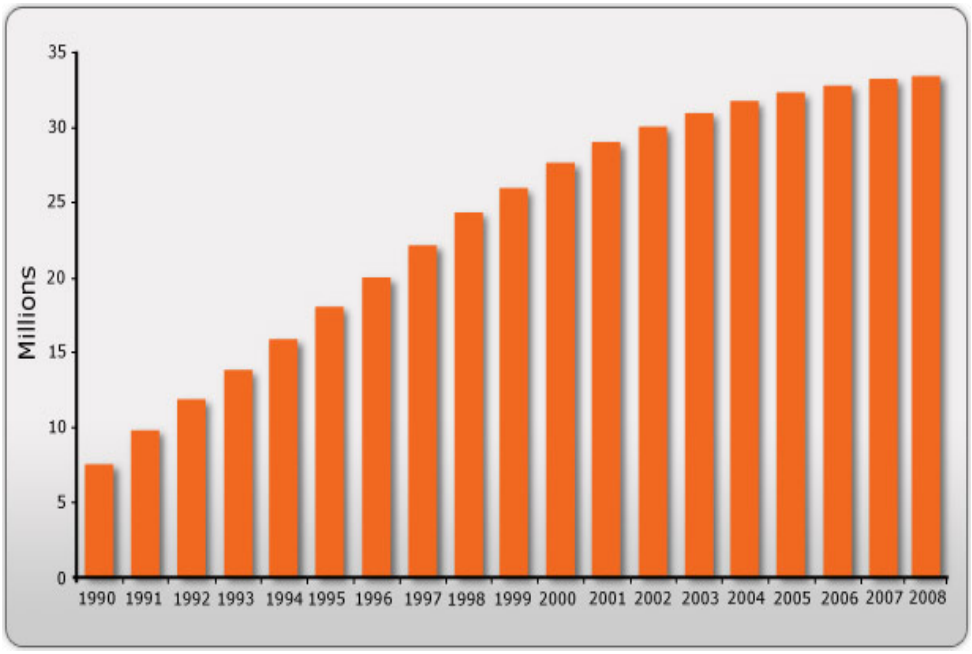
TABLE 1

Worldwide statistics of people living with HIV/AIDS

People Living with HIV/AIDS in 2008	Estimate (million)
Adults	31.3
Women	15.7
Children	2.1

TABLE 2

World HIV/AIDS statistics



European HIV and AIDS statistics:

According to UNAIDS estimates, around 2.3 million people were living with HIV in Europe at the end of 2008.[10] The number of AIDS cases per million populations has declined slightly in western and central Europe, as a result of the widespread availability of antiretroviral drugs in this region.[11]

South Africa National HIV survey 2008:

10.9% of all South Africans over 2 years old were living with HIV in 2008. Among females, HIV prevalence is highest in those between 25 and 29 years old; among males, the peak is in the group aged 30-34 years[12]. Antenatal surveillance is internationally recognized as the most useful way of assessing HIV prevalence among women aged 15-19 years old, for which the antenatal survey produce a rate much higher than the household survey (15.9% compared to 9.4%).[13]

Sub-Saharan Africa HIV and AIDS Statistics:

An estimated 22.4 million adults and children were living with HIV in Sub-Saharan Africa at the end of 2008. During that year an estimated 1.4 million Africans died from AIDS. Around 14.1 million children have lost one or both parents in the epidemic, and in 2008 an estimated 1.8 million children were living with HIV.[14]

India HIV and AIDS statistics:

India has a population of one billion, around half of whom are adults in the sexually active age group. The first AIDS case in India was detected in 1986[15], and since then HIV infection has been reported in all states and union territories. The highest HIV prevalence rates are found in Andra Pradesh, Maharashtra, Tamil Nadu and Karnataka in the south and Manipur and Nagaland in the North-East. In the south states, HIV is primarily spread through heterosexual contacts. Infections in north-east are mainly found among injecting drug users (IDUs) and sex workers[16]. The average HIV prevalence among women attending antenatal clinics in India is 0.48% much higher rates found among people attending STD clinics, female sex workers (5.1%), Injecting drug users (7.2%) and men who have sex with men (7.4%)[17]

Antiretroviral drugs (ARVs) which can significantly delay the progression from HIV to AIDS have been available in developed countries since 1996. In India an estimated 300,000 people were receiving free AVRs by January 2010. This represents less than half of those estimated to be in antiretroviral treatment in India.[18]

HIV TRANSMISSION

The primary routes of transmission are through sexual intercourse, vertically from mother to infant during pregnancy or early infancy, percutaneous exposure and exposure to infected blood and its products[29].

SEXUAL TRANSMISSION:

Sexual transmission of HIV infection can occur following vaginal and anal intercourse, and also through oral sex. Male to female HIV transmission is twice as effective as female to male transmission[19]. The risk of male to female of HIV transmission during a single genital exposure has been calculated as less than 0.2%[20]. The risk of male to male transmission per episode of unprotected anal intercourse has been estimated to between 0.3% and 10%.[21]

MOTHER TO CHILD TRANSMISSION (MTCT)

HIV infection from an HIV positive mother to her child can occur during pregnancy, delivery or breast feeding[22].

Without treatment around 15-30% of babies born to HIV positive women become infected with HIV during pregnancy and delivery[23]. A further 5-20% becomes infected through breast feeding[24]. In 2009, an estimated 4, 30,000 children become newly infected with, the majority of them through MTCT. [22]

The risk associated with prenatal transmission of HIV–1 are multifactorial. Known risk factor include high maternal plasma viremia, advanced clinical HIV–1 diseases, reduced maternal immunocompetence, vaginal delivery and a lengthy interval between rupture of the amniotic membrane and delivery. [23] In addition, exposure to maternal blood, presence of ulcerative genital infection in maternal vaginal track at the time of delivery, illicit drug use during pregnancy, prematurity and low birth weight have all been associated with increased mother to child transmission.[25]

There is evidence suggesting that pregnancy also favours the progression of the HIV disease in the mother. The most important determinant is the viral load present in the mother.[26]

BLOOD – BORN TRANISMISSION OF HIV:

There is approximately a 90% risk of transmission of HIV to a recipient if the blood is donated during the window period. Donor screening and HIV testing of donors can prevent HIV transmission through blood and blood products. [29]

OCCUPATIONAL EXPOSURE:

The risk of HIV infection from a single percutaneous exposure is estimated to be approximately0.3%. The risk of transmission following exposure to intact mucosa and skin is postulated to be considerably less.[27]

ORGAN AND TISSUE DONATION:

HIV transmission can occur following the transplantation of human organs or following bone marrow transplantation from infected donors.[28]

STAGES OF HIV INFECTION:

The HIV infected person will progress more or less sequentially through a series of characteristic clinical stages with the progressive depletion of CD_{4+} lymphocytes counts.[30]

The HIV Infection can be divided into four stages

1. Acute seroconversion illness or primary HIV infection
2. Asymptomatic stage of HIV infection
3. Late symptomatic stage of HIV infection.
4. Late Symptomatic and advanced stage of HIV infection (AIDS)

STAGE 1: PRIMARY HIV INFECTION

Primary infection is also called the seroconversion illness or acute HIV infection.[30] However 30% - 50% of individuals present with mononucleosis like illness within 2 -6 week of exposure,[31] resolves within 2 weeks. Transient CD4+ lymphopaenia has been seen in the few patients studies.[32] It has been observed that a longer duration of symptomatic seroconversion is associated with more rapid progression of the disease.[33, 34] This being a stage of acute viremia, high levels of circulating free virions can be detected by RT- PCR.[34] The tests for HIV antibodies are negative in

this stage.[30] A seroconversion illness associated with HIV – 2 infections has been reported.[35]

STAGE 2: ASYMPTOMATIC STAGE OF HIV INFECTION:

Duration varies from 6 months to 15 years. [31]

During this stage patient is asymptomatic and generally has no findings on physical examination excerpt for persistent generalized lymphadenopathy (PGL) in some cases.[36] Occasionally people may experience headaches, which can be recurrent or chronic debilitating in nature.[37] The rate of decline of the CD_4 cell count has been estimated as 84 cells per year for the first 4 years after seroconversion.[38]

STAGE 3: EARLY SYMPTOMATIC STAGE OF HIV INFECTION:

Using statistical models, it has been projected that 76% of HIV infected people will develop late symptomatic diseases by the year 16 of infection.[39] The rate of HIV 1 disease progression has been estimated to be 3 to 4 times greater than that of HIV 2.[40] Though in the early stage of HIV disease, the levels of virus detectable in the peripheral blood often remain low.[30] However this masks every rapid rate of virus production and destruction in lymphoid tissue.[41]

Anergy to skin testing increases as the CD4+ count falls below 400/µl with obvious implications for tuberculosis screening. [30] Common episodic

conditions during this stage of disease include Herpes zoster, Candidiasis, Seborrhoeic dermatitis, skin and nail infections like impetigo, folliculitis, fungal intertrigo and paronychia along with bacterial infection like pneumonia, bronchitis and sinusitis.[30]

Recurrent Herpes zoster is one of the earliest manifestation of symptomatic disease. Oral candidiasis and Oral hairy leukoplakia (OHL) also occur in Stage 3 of HIV[30]. In one study of cohorts of homosexual men, the 2 years progression of AIDS for men with Herpes zoster, Candidiasis, Oral hairy leukoplakia and constitutional symptoms were 25%, 39%, 42% and 100% respectively.[42]

STAGE 4: LATE SYMPTOMATIC AND ADVANCED STAGES OF HIV INFECTION (AIDS)

In the absence of treatment the HIV infection progress and the manifestation of late symptomatic disease typically appears in CD_{4+} count fall below 200/µl. In the absence of prophylaxis, Pneumocystis carinii, pneumonia (PCP) is the most common and life threatening infection[30].

Crowe found that the order of appearance of opportunistic infection and malignancies during this stage was strongly correlated with the CD4+ lymphocyte count[43].

In advanced stage of HIV disease, where CD4+ cell counts have fallen below 50/µl, chorioretinits, mycobacterium avium complex (MAC) and primary cerebral lymphoma may occur and HIV-1 associated dementia appears to progress more rapidly. Infections such as MAC, CMV, strongyloides stercoralis, herpes zoster or tuberculosis, that normally colonise superficially or are limited to an organ system or a local anatomic region, may invade other tissue or disseminate widely[30]. Several of these opportunistic diseases have been described in HIV- 2 infected individuals.

MUCO CUTANEOUS MANIFESTATIONS OF HIV INFECTION:

Dermatological manifestations are seen at every stage of HIV infection and are often its presenting features[44]. The most common mucocutaneous lesions are oro-esophageal candidiasis, oral hairy leukoplakia, Kaposi's sarcoma, histoplasmosis, and, in Asia, *Penicillium marneffei* infection. Non-HIV-related skin lesions, such as psoriasis, seborrhoeic dermatitis, and nodular prurigo, may be the initial presentation among HIV infected patients attending outpatient clinics.[45]

PATHOGENESIS

Since HIV is not eliminated after primary infection, persistent virus replication occurs in lymphoid organs during the course of HIV infection.[46] Destruction of lymphoid tissue results in severe impairment of the ability to

maintain an effective on going HIV specific immune response and to generate immune response against new pathogens.[47] Epidermal Langerhans cells may become infected by HIV, and decreased Langerhans cell function could account for some of the cutaneous manifestations of HIV disease.[46]

CUTANEOUS MANIFESTATIONS OF HIV

CLASSIFICATION OF DERMATOSES IN HIV /AIDS:

FUNGAL INFECTIONS

Candidiasis

Pityrosporum infection

Dermatophytosis

Cryptococcosis

Histoplasmosis

Penicilliosis

Aspergillosis.

VIRAL INFECTIONS

Herpes simplex infection

Varicella zoster infection

Epstein Barr virus infection

Cytomegalovirus infection

Human papilloma virus infection

Molluscum contagiosum

Hepatitis B and hepatitis C virus Infection

BACTERIAL INFECTIONS

Staphylococcal infections

Mycobacterial infections

Bacillary angiomatosis

Lymphogranuloma venereum

Syphilis

Chancroid

PARASITIC INFECTIONS

Scabies

Demodicidosis

INFLAMMATORY DISODERS

Seborrhoeic dermatitis

Psoriasis

Reiter's disease

Ichthyosiform dermatoses.

Papular and follicular eruption of HIV

Drug reaction

NEOPLASIA

Kaposi sarcoma

Lymphoma

Other cutaneous carcinomas

MISCELLANEOUS DERMATOSES

Hidradenitis suppurativa

Generalized hyper pigmentation

Insect bite reactions --- increased

Photosensitive eruptions

HAIR CHANGES

NAIL CHANGES

CLASSIFICATION OF DERMATOSES IN HIV/ AIDS:
FUNGAL INFECTIONS

Candidiasis

Candidiasis or **thrush** is a fungal infection (mycosis) of any of the *Candida* species (all yeasts), of which *Candida albicans* is the most common.[48, 49] Mucocutaneous candidiasis occurs in 3 forms in persons with HIV infection: oropharyngeal, esophageal, and vulvovaginal disease. Oropharyngeal candidiasis (OPC) was among the initial manifestations of HIV-induced immunodeficiency to be recognized[50,51] and typically affects the majority of persons with advanced untreated HIV infection. Presenting months or years before more severe opportunistic illnesses, OPC may be a sentinel event indicating the presence or progression of HIV disease.[52,53,54] Although usually not associated with severe morbidity, OPC can be clinically significant. Severe OPC can interfere with the administration of medications and adequate nutritional intake, and may spread to the esophagus.[55] Esophageal candidiasis remains one of the most common opportunistic infections in countries where combination antiretroviral therapy (ART) is a routine part of the standard of care.[56] Vulvovaginal candidiasis is an important concern for women with HIV infection, although the relationship of vulvovaginal candidiasis to HIV infection remains unclear.[57] In resource-poor nations, mucocutaneous candidiasis is a formidable problem.[58,59] Despite the frequency of mucosal disease,

disseminated or invasive infections with *Candida* and related yeasts are surprisingly uncommon.

Pityrosporum infection

Malassezia furfur has been linked to several skin diseases, including seborrhoeic dermatitis, folliculitis, pityriasis versicolor, and atopic dermatitis.[60] Seborrheic dermatitis is one of the earliest clinical markers of HIV infection.[47] *Pityrosporum* folliculitis (PF) is an inflammatory skin disorder that typically manifests as a pruritic, follicular papulopustular eruption distributed on the upper trunk of young to middle-aged adults. Weary et al first described *Pityrosporum* folliculitis in 1969, and, later in 1973.[61] *Pityrosporum* folliculitis is present on body locations in which *Malassezia* organisms are most abundant: back and chest, neck, shoulders, scalp.[62]

Dermatophytosis

The dermatophyte infections that HIV-infected patients develop tend to be more severe, atypical and refractory to treatment.[63] Normally, these fungal infections remain superficial in the epidermis, but in HIV patients the infection can become invasive. The most common presentation of deep or locally invasive fungal infection is the eruption of nodules near the initial site of infection, but abscesses, mycetomas and atypical lesions are also possible. *T. rubrum* is most often the cause of this deeper infections.[64]

Cryptococcosis

Cryptococcus neoformans is abundant in soil contaminated by pigeon droppings. Cryptococcosis is the most common cause of HIV-related meningitis in Central and Southern Africa, accounting for up to 40% of cases.[65] Pulmonary cryptococcosis is also being increasingly recognized with one study reporting 7% prevalence in a group of South African miners (HIV prevalence of 24%).[66] Secondary cutaneous infections occur in up to 15% of patients with disseminated cryptococcosis and often indicate a poor prognosis. Lesions usually begin as small papules that subsequently ulcerate, but may also present as abscesses, erythematous nodules, or cellulitis.[67]

Histoplasmosis

Histoplasmosis is the most common of the endemic mycoses in patients with AIDS.[68] Disseminated histoplasmosis (DH) initially was reported in patients with AIDS in 1982.[69] In patients with advanced HIV infection, histoplasmosis almost always is manifested by signs of progressive disseminated disease, as opposed to the asymptomatic or limited pulmonary infection observed in the majority of healthy individuals exposed to *H.capsulatum*.[70] Histoplasmosis is common among AIDS patients because of their depressed immune system.[71] Skin manifestations are protean, ranging from papules to ulcers to erythema multiforme.[72]

Penicilliosis

Penicilliosis is an infection caused by *Penicillium marneffei*, a dimorphic fungus endemic to Southeast Asia and the southern part of China.[73] Persons affected by penicilliosis usually have AIDS with low CD4+ lymphocyte count, typically <100 cells/μL. The average CD4+ count at presentation is 63.5 cells/μL.[74, 75] The most common presentation is disseminated infection manifested by fever, skin lesions, anemia, generalized lymphadenopathy, and hepatomegaly.[74] Patients with penicilliosis have a poor prognosis without treatment.[74] Skin lesions are present in approximately two third of cases and can be varied in appearance. Generalized papular eruptions, central umbilicated papules resembling those of molluscum contagiosum, acne like lesions and folliculitis all may occur. Skin lesions commonly occur on the face, trunk, and extremities. Pharyngeal and palatal lesions also can be seen.[76] Subcutaneous nodules occasionally can be seen.[74]

Aspergillosis

Cutaneous *Aspergillosis* can occur as primary or secondary infection. Primary cutaneous aspergillosis is associated with local skin injury (from tape, intravenous catheter sites) and neutropenia. Lesions can appear as erythematous indurations with overlying pustules, hemorrhagic ulcers, or molluscum contagiosum-like lesions. Despite the severe immunosuppression that results from advanced HIV infection, there are relatively few cases of aspergillosis in patients with HIV disease.[77]

VIRAL INFECTIONS

Herpes Simplex Infection

Among HIV-1 infected individuals, HSV-1 and HSV-2 infections are common. In recent years, a number of studies have focused on the prevalence of HSV-2 among HIV-1 infected individuals, finding seroprevalence of 50-90% in some populations, significantly higher than among those without HIV-1.[78] HIV-1-infected persons, however, also can have frequent or persistent HSV lesions, often with extensive or deep ulcerations, particularly among those with low CD4+ count. In one study, the frequency of genital ulcer disease consistent with reactivation of genital herpes was found to increase in a stepwise fashion with declining CD4+ count.[79] Among HIV-1-infected persons, HSV mucosal shedding occurs

more frequently, and with higher quantity of HSV, among those with lower CD4+ counts.[80, 81, 82]

Varicella Zoster Infection

Varicella zoster virus (VZV) infection is commonly seen early in the course of HIV infection, particularly in healthy-appearing individuals, before the onset of other symptoms.[83,84,85,86,87] Because most HIV-infected persons have had varicella previously, the initial manifestation of VZV infection is usually Herpes zoster. During the course of HIV disease, herpes zoster often precedes thrush and oral hairy leukoplakia by about one year.[85] Unlike zoster in individuals without HIV infection, this dermatomal eruption may be particularly bullous, hemorrhagic, necrotic, and painful in HIV-infected persons. The duration of blisters and crusts is usually 2 or 3 weeks. The approximate duration of significant pain is also 2 or 3 weeks. Necrotic lesions may last for up to 6 weeks and heal with severe scarring. This dermatomal scarring is characteristic of HIV-infected patients and should be sought when evaluating at-risk individuals.[88] Recurrences have been reported in up to 25% of African HIV-infected persons with herpes zoster.[86]

Epstein Barr Virus Infection

It is a gamma herpesvirus, also called human herpesvirus 4.[89,90] Oral hairy leukoplakia is a hyperplastic EBV-induced mucocutaneous epithelial cell disease and the first pathologic manifestation attributable to replicative

EBV infection,[91] making the lesion unique in EBV biology. It is seen in up to 25% of homosexual men with AIDS but has been reported as well in other immunocompromised patients and, rarely, healthy individuals.[91, 92]

Cytomegalovirus Infection

In humans it is commonly known as Human Herpesvirus[93]. Most healthy people who are infected by CMV after birth have no symptoms.[93] Some of them develop an infectious mononucleosis/glandular fever-like syndrome[94].

Human Papilloma Virus Infection

Superficial cutaneous infection with human papilloma virus (HPV) or warts, occurs with increased frequency in immunosuppressed patients. Lesions may be extensive and resistant to therapy. Condyloma acuminata are of special significance in persons with HIV infection. Cervical dysplasia and carcinoma are clearly associated with HPV infection.[95] Unfortunately, anorectal warts in HIV-infected men are difficult to eradicate.[96] Female HIV-infected patients must have regular gynaecological examinations and Papanicolaou (Pap) smears.[97, 98]

Molluscum Contagiosum

Molluscum Contagiosum is characterised by a faint whitish core at the center of each papule, some of which may be slightly umbilicated.

Molluscum contagiosum occurs in approximately 10 to 20% of HIV-infected persons.[83] Early in the infection, the lesions are usually mild and localized to the groin or face. Once CD4 counts fall below 200, however, lesions tend to proliferate. They often number greater than 100 and may involve the face, trunk, and groin; there is a predilection for the eyelids.[99] Extensive molluscum contagiosum is a cutaneous marker of advanced HIV disease (CD4 < 50). At this stage, molluscum may extend onto mucosal surfaces of the lips or conjunctiva. Patients with advanced HIV disease are rarely cured of their molluscum. The finding of subclinical, microscopic infection in apparently normal skin may explain failure to cure patients.[100] Giant mollusum contagiosum should make one to suspect HIV disease.

Hepatitis B Virus Infection

HBV is transmitted primarily through sexual contact and intra venous drug use, whereas perinatal and early childhood exposures are responsible for the majority of HBV transmission in high-prevalence countries. Although risk factors are similar, HBV is transmitted more efficiently than HIV. [101, 102, 103] Up to 90% of HIV-infected persons have at least one serum marker of previous exposure to HBV.[104, 105] And approximately 10% have evidence of chronic hepatitis B.[106, 107, 108]

Hepatitis C Virus Infection

There are about 150 million chronic hepatitis C virus (HCV) carriers throughout the world, with an estimated global prevalence of 3% (range 0.1-5%). [109] Following exposure to HCV, approximately 85% of patients develop chronic infection.[110, 111] Co infection with HCV in HIV-infected individuals is common, presumably due to the shared route of transmission of these viruses. The prevalence of HCV infection among all HIV-infected individuals can be as high as 40% but this prevalence varies substantially among different risk groups.[112] The prevalence of HCV among intra venous drug users (IDUs) who are HIV infected is 50-90%.[113,114,115,116,117] In a large cohort of 3,048 HIV-infected subjects from the EuroSIDA study, 33% were HCV antibody positive and more than 75% of IDUs in this population were coinfected.[118] However, the prevalence of HCV in HIV-infected men who have sex with men (MSM) is similar to that observed in HIV-negative homosexual males at 4-8%.[115,119] Although the rate of sexual transmission of HCV is low (<5%), this rate may be increased in the presence of HIV. [116,120,121,122,123]

The rate of mother-to-infant transmission of HCV increases in the presence of HIV, presumably due to high levels of HCV viremia observed in co infected individual.[124, 125,126] HIV co infection has been associated with a

more rapid progression of liver disease as well as a higher prevalence of cirrhosis.[115, 127,128,129]

BACTERIAL INFECTIONS

Staphylococcal Infections

Staphylococcus aureus is the most common cutaneous bacterial infection in persons with HIV disease. Approximately 50% of HIV-infected persons are nasal carriers of S. aureus, explaining in part the high rate of infection.[130, 131] Infection with *S. aureus* may occur before any other signs or symptoms of HIV infection. Morphologic patterns that may occur include: bullous impetigo, ecthyma, folliculitis, hidradenitis-like plaques, abscesses, cellulitis, and pyomyositis.[132]

Bullous Impetigo

Bullous impetigo is common in hot, humid weather, presenting as very superficial blisters or erosions, most commonly seen in the groin or axilla. Because the blisters are flaccid, they are short-lived; often only erosions or yellow crusts are present. These lesions closely mimic cutaneous candidiasis.[133]

Ecthyma

Ecthyma is an eroded or superficially ulcerated lesion with an adherent crust. Under this crust is often a plane of purulent material teeming

with staphylococci. Removal of this crust is necessary to treat the lesion topically.

Folliculitis

Folliculitis due to *S. aureus* occurs most commonly in the hairy areas of the trunk, groin, axilla, or face, especially in men who shave. Follicular pustules are the primary lesion. Often the follicular lesions of the trunk are intensely pruritic and may be mistaken for other pruritic dermatoses, such as scabies.[133] About 50% of HIV-infected persons with scabies have coexistent *S. aureus* folliculitis. Occasionally, follicular lesions extend more deeply, forming abscesses. Rarely, all follicles across several square centimeters are infected, forming a large, violaceous, hidradenitis-like plaque. The plaque may be studded with pustules and have deep tracts connecting infected follicles. These plaques may mimic Kaposi's Sarcoma, but overlying pustules are quite unusual in KS. Rarely, abscess of the muscle (pyomyositis) may occur.[132, 133,134,135,136]

Mycobacterial infections

The World Health Organization (WHO) estimates that Tuberculosis is the cause of death for 13% of persons with AIDS.[137] Tuberculosis infection occurs when a susceptible person inhales droplet nuclei containing *Mycobacterium tuberculosis* organisms, generated when persons with pulmonary or laryngeal Tuberculosis disease cough, sneeze, shout, or sing

[138]. Persons with latent Tuberculosis infection are, by definition, asymptomatic. Among HIV-infected persons, the presentation of active TB disease is influenced by the degree of immunodeficiency. [137,138]

In addition, early after initiating ART in severely immunosuppressed patients, previously unrecognized subclinical TB can be unmasked by reconstitution of the immune system.[139,140] In HIV-infected patients without pronounced immunodeficiency (e.g., $CD4^+$ count >350 cells/μL), HIV-related TB clinically resembles TB among HIV-uninfected persons. The majority of patients have disease limited to the lungs, and common chest radiographic manifestations include upper lobe fibronodular infiltrates with or without cavitation[141]. However, extrapulmonary disease is more common in HIV-infected persons than in HIV-uninfected persons, regardless of CD4+ counts, although clinical manifestations are not substantially different from those described in HIV-uninfected persons. TB must be ruled out in diseases of every organ [142], but especially those related to CNS or meningeal symptoms in which early TB treatment is essential to improve the outcome.[143, 144]

In patients with CD4+ count <200 cells/μL, Tuberculosis can be a severe systemic disease with high fevers, rapid progression, and sepsis syndrome.[145]

In patients with AIDS, however, it Mycobacterium avium complex (MAC) is one of the most common serious opportunistic infections (OIs). Among HIV-infected individuals, disseminated MAC historically has occurred almost exclusively in patients with a CD4 count <50 cells/μL.[146, 147] Colonization of the respiratory or gastrointestinal (GI) tract by MAC can occur without evident morbidity; however, MAC colonization of these sites indicates that patients are at increased risk for developing disseminated MAC infection.[148] Combination antiretroviral therapy (ART) has been associated with reduction in AIDS-related mortality, days of hospitalization, and the incidence of new OIs.[149] However, there have been numerous reports of aberrant clinical presentations of MAC since the introduction of combination ART.[150, 151,152,153,154,155]

Bacillary angiomatosis

Bacillary angiomatosis (BA) is a disease characterized by unique vascular lesions caused by infection with small, gram-negative organisms of the genus *Bartonella*. Virtually all patients with this disease are infected with HIV1. A major challenge in the diagnosis of cutaneous BA is the diverse presentation of lesions.[156, 157] Skin lesions may be classified as predominantly cutaneous or subcutaneous.[158] Cutaneous lesions are often papular and red with a smooth or eroded surface,[159,160,161,162,163,164,165,166,157,167,168,169,170,171] and papules may enlarge to

form large, pedunculated lesions.[159,157,175] These lesions have an obvious vascular appearance and an erythematous base and bleed profusely when traumatized. Other papular lesions and plaques may be dusky in colour, dry, scaly, and hyperkeratotic, with minimal clinical evidence of vascularity.[156] Cutaneous BA also may have the appearance of a cellulitic plaque; these cutaneous lesions often overlie osteolytic lesions.[156, 162] Dark-skinned patients may develop an indurated, hyper pigmented plaque that may be nearly black.[176] Cutaneous lesions may occur singly or number in the hundreds. [157,159,164,175]

Subcutaneous lesions may present as one or more deep nodules with flesh-colored[157] or erythematous overlying skin.[176, 177] Reports also describe presentation as a single, deep soft-tissue mass with normal-appearing overlying skin. [156,178,179]

Lymphogranuloma venereum

Lymphogranuloma venereum (LGV) is caused by *Chlamydia trachomatis* serovars L1, L2, or L3.[180] The most common clinical manifestation of LGV among heterosexuals is tender inguinal and/or femoral lymphadenopathy that is typically unilateral. This infection is found most frequently in tropical and subtropical areas of the world.[181] The diagnosis is difficult to establish on clinical grounds alone and frequently relies upon

either serologic testing, culture, or more recently, nucleic acid amplification testing of direct specimens.[182, 183]

Syphilis

Syphilis is the result of a bacterial infection of the genital tract by the bacterium Treponema pallidum. Most HIV-infected patients with *T pallidum* infection present with typical dermatologic clinical features of primary and secondary disease, such as chancres and diffuse maculopapular rashes.[184, 185, 186, 187, 188] Syphilis can present in one of four different stages: primary, secondary, latent, and tertiary.[189] Syphilis is associated with increased risk for HIV sexual acquisition and transmission.[190, 191] Early syphilis in HIV-infected persons may also cause a transient decrease in CD4+ count and increase in HIV viral load that improves with standard treatment regimens.[192, 193, 194]

Chancroid

Chancroid, caused by infection with *Haemophilus ducreyi*, is characterised by ano-genital ulceration and lymphadenitis with progression to bubo formation. The incubation period for this disease is short, around 3–10 days, and the initial lesion is a papule that may progress to form an ulcer through an intermediate pustular stage.[197] In fact, HIV has been detected in chancroidal ulcers.[195,196]

PARASITIC INFECTIONS

Scabies

Scabies in HIV-infected persons usually presents with the typical pattern of pruritic papules with accentuation in the intertriginous areas, genitalia, and finger webs. With advancing immunosuppression, the infestation may exaggerate, becoming more widespread and refractory to treatment, and sparing the characteristic areas.[198] In rare cases, true crusted (Norwegian) scabies may occur in patients with advanced HIV disease. Norwegian scabies is nonpruritic and appears as thick crusts over some areas of the body. These crusts teem with mites and are highly contagious.[199, 200,201,202]

Demodicidosis

Mites such as Demodex folliculorum and D. Brevis are natural host of the human pilosebaceous follicle and are responsible for skin disorders like , papular and pustular eruptions of the scalp, acne rosacea etc. . The highest concentration of the demodex is found in the cutaneous areas with more numerous sebaceous glands. Demodicidosis in patients infected with HIV is already reported in the relevant literature both in adults and in children.[203]

INFLAMMATORY DISODERS

Seborrhoeic dermatitis

Seborrhoeic dermatitis is a mild eruption, usually affecting the scalp and central areas of the face, which occurs in up to 5% of the non-HIV-infected population. Early in the HIV epidemic, many patients with symptomatic HIV disease had particularly severe seborrheic dermatitis. One study found an 83% point prevalence of seborrheic dermatitis associated with advanced HIV disease and a 42% point prevalence with symptomatic HIV disease.[204]

Psoriasis

The clinical appearance of psoriasis is similar in HIV-infected and no infected individuals. The incidence of severe involvement of the axillae and groin (sebopsoriasis), however, is increased in HIV-infected patients.[205] In addition, psoriatic erythroderma is not rare in these patients.[206] As in routine psoriasis, pruritus may be a serious problem for the HIV-infected patient with psoriasis. With scratching, secondary infection of excoriated psoriatic plaques with *S. aureus* may occur. Erythrodermic psoriasis in HIV-infected patients may be a sign of *S. aureus* septicemia, and the psoriasis may improve dramatically with only intravenous antibiotics.[207]

Reiter's disease

Classic Reiter's disease consists of the triad of arthritis, conjunctivitis, and urethritis. Usually, symptoms of HIV infection and Reiter's disease

appear simultaneously or the HIV infection is symptomatic before the onset of Reiter's disease. Patients may initially present, however, with classic cutaneous lesions of RS before manifesting clinical symptoms of HIV infection.[206, 208]

Ichthyosiform dermatoses

HIV-infected patients commonly complain of increasing dryness of the skin (xerosis).[209,210] Patients with an atopic diathesis (hay fever, asthma, or previous atopic dermatitis) are predisposed to this type of xerotic eczema. Dry and thickened skin, as is seen in acquired ichthyosis, is seen in patients with advanced HIV disease. Thickening of the palms and soles may be present as well.[211,212]

Papular and follicular eruption of HIV

Pruritic Papular Eruptions

Pruritic papules are common in HIV infection[213] and are due to various causes. Reports describe *S. aureus* folliculitis, eosinophilic folliculitis, demodicidosis mites,[214] insect bite reactions, and granulomas with no identifiable infectious agent (e.g., granuloma annulare[215]) as causes of itching in the setting of HIV. We think there is no specific and unique "papular eruption of HIV infection." Most patients with this diagnosis have eosinophilic folliculitis[216]. Rarely, pruritus with no primary skin lesions may be the presenting sign of HIV disease.[217]

Eosinophilic Folliculitis of HIV Disease

Bacteria do not cause all folliculitis in HIV-infected patients. Culture-negative folliculitis commonly reveals eosinophils on biopsy.[218] Cases in which the biopsy shows eosinophils within or around the follicle have been called eosinophilic folliculitis of HIV infection. This disorder typically occurs in HIV-infected persons with helper T cell counts below 200, so it is an important cutaneous marker of a specific stage of HIV disease. The eruption waxes and wanes. Intensely pruritic, edematous, urticarial papules and pustules appear in crops on the trunk or face or both. Cultures and histological examination for infectious agents are negative, but a relative peripheral eosinophilia may be present.[219, 220]

Drug reaction

Trimethoprim Sulfamethoxazole is used frequently in managing PCP. The incidence of adverse reactions to the drug is very high; Most reactions occur in the second week of therapy and are typical maculopapular/morbilliform reactions. They begin in the groin and pressure areas and quickly generalize.[221] Other drug-induced hypersensitivity reactions in HIV-infected patients are urticarial reactions, exfoliative erythroderma, fixed-drug eruption, erythema multiforme, and toxic epidermal necrolysis. These reactions are most often due to antibiotics,

especially TMP-SMZ and the penicillins. These reactions may take up to 8 weeks to clear. Multiple sequential reactions are not unusual.[222]

NEOPLASIA

Kaposi's sarcoma

Kaposi's sarcoma (KS) is a neoplasm of endothelial cells involving the skin and, at times, other internal organs. KS is common among HIV-infected persons, but there is not an equal incidence in all risk groups. Most KS patients are homosexual men. In one series, 46% of homosexual men with advanced HIV disease had KS at the time of their initial diagnosis. The incidence in heterosexual injection drug users is only 3.8%.[223] Several homosexual men have been identified who developed KS but who are uninfected with HIV by all current testing methods.[224] These data support the involvement of a sexually transmitted infectious agent. Herpes virus 8 (HHV-8) has been associated with KS.[225, 226,227,228,229,230,231]

KS may affect any portion of the cutaneous surface. Initially, it appears as red-to-brown flat macules. Papules, nodules, and tumors may also be present or develop later. KS may affect mucosal surfaces and internal organs with or without involving the skin. Visceral involvement occurs in 72% of patients with advanced HIV disease and KS, most often affecting the gastrointestinal tract (50%), lymph nodes (50%), and lungs (37%).[232] The natural history of HIV-associated KS is not uniform, but the prognosis is

poor. The average survival of patients is 18 months.[232] Most individuals have generalized, slowly progressive disease; others have stable KS. Even more rarely, the disease may resolve spontaneously.[233] Poor prognostic findings include generalized disease and coexistent opportunistic infections; the latter are the most common cause of death.[232]

Lymphoma

HIV-1-associated lymphoma was first incorporated into the U.S. Centers for Disease Control and Prevention's (CDC) case definition of AIDS in 1985.[234,235] Without effective antiretroviral therapy, it is estimated that 5-10% of all HIV-infected individuals will have lymphoma as either an initial or subsequent AIDS-defining condition.[236] A 1991 review[237] of 2,500 cases of HIV-1-associated lymphomas revealed that approximately 80% arose in the periphery (i.e., "systemic" lymphomas), and 20% arose in the central nervous system (CNS).[238,239,240] For HIV-1-associated systemic lymphomas, widespread disease involving extranodal sites is common.[241, 242,243]

Other cutaneous carcinomas

Other cutaneous carcinomas are not unusual in HIV-infected patients. Fair-skinned patients with significant prior sun exposure are at risk for basal cell carcinomas (BCCs) and squamous cell carcinomas.[244, 245,246] BCCs commonly present on the back or chest as nonhealing scaly patches that may erode or bleed. Squamous cell carcinomas are associated with more sun exposure and are more common on the head and neck. Occasionally, HIV-

associated lymphoma of either T or B cells will present in the skin.[246, 247,248,249]

MISCELLENEOUS DERMATOSES

Hidradenitis suppurativa

Hidradenitis suppurativa (HS) is a disorder of the terminal follicular epithelium in the apocrine gland-bearing skin. It is characterized by comedo-like follicular occlusion, chronic relapsing inflammation, mucopurulent discharge, and progressive scarring.[250] The disease begins with the obstruction of the apocrine gland duct, resulting in the infection of the retained secretions.[251] Atypical sites were involved in HIV patients such as face, thighs which could be due to HIV-related immunosuppression. Etiology may also have an endocrinal component, and HIV-associated endocrinopathies may alter the course of the disease. Association of AIDS with a chronic skin condition like HS is a therapeutic challenge.[252]

Generalised hyper pigmentation

Generalised hyper pigmentation and addisonian pigmentation, apparently independent of drug therapy have been reported in AIDS patients in southeast Asia.[253,254] Numerous factors are responsible, including drugs for treatment and prophylaxis, endocrine disturbances caused by HI, sun exposure and OIs.[253]

Insect bite reactions --- increased

Urticarial pruritic papules are a common morphologic lesion in HIV-infected patients. In occasional patients, this lesion is associated with insect bites and is called papular urticaria. In HIV-infected patients in San Francisco, except for scabies mites, fleas most commonly cause papular urticaria. In the southern United States, mosquitoes may be the primary offender.[255, 256]

Photosensitive eruptions

Reports rarely describe photosensitivity in HIV-infected persons.[257] Such reactions are not unusual, however. Erythematous patches and plaques appear during periods of increased sun exposure, primarily on exposed body parts, especially the dorsa of the hands, extensor forearms, side of the neck, and face. The photosensitivity is usually due to the shorter ultraviolet spectrum (UVB). Frequently prescribed photosensitizing medications (nonsteroidal anti-inflammatory drugs and TMP-SMZ) used in HIV-infected patients may play a role in their photosensitivity.[88]

HAIR CHANGES

Severe tinea capitis with significant hair loss has been reported in several adult patients with AIDS[258,259].Changes in hair length and consistency[260,261], hypertrichosis of eyelashes and very long

eyelashes[262,263],alopecia areata[264,265] and premature graying of the hair[266,267] have been reported in association with HIV infection.

NAIL CHANGES

Diseases of the nail may be observed in up to32% of patients.[259] Proximal subungual onychomycosis appear to be related to the effect of HIV infection on immune system.[259, 268] Others such as nail dyschromia are the result of antiretroviral therapy.[259]

MAJOR MUCOCUTANEOUS FINDINGS IN INDIAN STUDIES

Criton et al studied the various mucocutaneous manifestations in HIV patients and found out that Generalised itching and dry skin is the commonest one followed by in order of frequency Lustreless hair, Hyper pigmented nail bed, Acne vulgaris, Oral candidiasis, Hyper pigmentation, Seborrhoea like lesions, Exaggerated insect bite reaction, Dermatophytosis and Viral infections.[269]

Muhlemann et al studied the various dermatological manifestations in HIV patients and found out that Extensive fungal infection is the most common, followed by Acneiform folliculitis, Seborrhoeic dermatitis, Axillary folliculitis, Kaposi's sarcoma, Xeroderma and Beard and neck impetigo. They concluded that chronic acneiform folliculitis, florid Beard and neck impetigo and fungal infections are early warning signs of AIDS.[270]

Goodman et al studied the various dermatological manifestations in HIV patients and found out that Candidiasis is the commonest one. The other manifestations are recorded in decreasing order Seborrhoeic dermatitis, Dermatophytosis, Xerosis/acquired ichthyosis, Herpes simplex, Molluscum contagiosum, Verrucae, Acne, Kaposi's sarcoma and Herpes zoster. They concluded that skin is the early and sensitive marker for the presence of clinically apparent HIV infecton.[271]

Matis et al studied the various mucocutaneous manifestations in HIV patients and found out that Onychomycosis is the commonest one followed by Seborrhoeic dermatitis, Oral hairy leukoplakia, Condyloma accuminata, Oral candidiasis, Herpes simplex, Molluscum contagiosum and Verruca vulgaris in decreasing order of frequency.[272]

Valle studied the various dermatological manifestations in HIV patients and found out that Oral candidiasis is the commonest one, followed by Seborrhoeic dermatitis, Tinea interdigitale, Asteatosis, Infective eczematoid dermatitis, Warts, Acquired Ichthyosis and Herpes simplex. They found out that gradual worsening of seborrheic dermatitis paralleled the deterioration of the clinical condition and the development of acquired ichthyosis and gradual worsening of asteatosis was related to progression from ARC to AIDS.[273]

Alesi et al studied the various mucocutaneous manifestations in HIV patients and found out that Seborrhoeic dermatitis is the commonest one, followed by Herpes simplex, Warts/ Condyloma accuminata, Oral hairy leukoplakia, Oral candidiasis,Folliculitis, Dermatophytosis, Kaposi's sarcoma and Herpes zoster in decreasing order of frequency. They concluded that Oral candidiasis and Herpes zoster are signs of transition to AIDS and Kaposi's sarcoma chronic Herpes simplex are classic signs of fully developed AIDS.[274]

Kar et al studied the various mucocutaneous manifestations in HIV patients and found out that Oral thrush followed by Herpes zoster, Seborrhoeic dermatitis, Monilial balanoposthitis, Eosinophilic folliculitis and Dryness of the skin in decreasing order of frequency. They concluded that fungal or viral infections were the largest category of cutaneous disorders associated with HIV infection.[275]

Kumarasamy et al studied the various mucocutaneous manifestations in HIV patients and found out that Oral candidiasis is the commonest finding. Other findings in the decreasing order of frequency are Multidermatomal Herpes zoster, Dermatophytosis, Genital herpes, Papular pruritic dermatitis, Staphylococcal infection, Oral hairy leukoplakia, Molluscum contagiosum, Genital warts and Scabies.[276]

Jindal et al studied the various mucocutaneous manifestations in HIV patients and found out that Herpes zoster is the commonest one, followed by Mucocutaneous candidiasis, Dermatophytosis, Molluscum contagiosum, Scabies, Pyoderma, Venereal warts, Genital herpes, Chancroid, Candidal balanoposthitis, Herpes labialis, Seborrhoeic dermatitis ,Pruritic papular eruptions, Mucosal lichen planus, Miliaria rubra, Lichen simplex chronicus and Drug rash.[277]

Aim of the Study

AIM OF THE STUDY

1. To collect data and analyze the dermatological presentations in HIV patients.

2. If the patients were on antiretroviral therapy to assess the frequency of specific dermatological diseases or adverse reactions to antiretroviral drugs.

3. To assess the common dermatological diseases in HIV patients.

4. To correlate the CD4+ count at which usually each dermatological disease will occur.

Materials & Methods

MATERIALS AND METHODS

The study was conducted in Department of Dermatology, Venereology and Leprology, Coimbatore Medical College Hospital during the period May2009 to April 2010. All the patients attending the Dermatology outpatient department at Coimbatore Medical College Hospital were screened and HIV patients with Mucocutaneous manifestations were enrolled.

Inclusion criteria:

All HIV patients attending the Skin and Venereology Department in Coimbatore medical college, age in between 18-55 years.

Exclusion criteria:

1. Patients less than 18 years and more than 55 years.
2. Pregnant woman.

All the HIV patients with mucocutaneous manifestations were enrolled in a chronological order. For each patient name, age, sex, address, occupation and income etc noted. Complaints, duration, history of recurrences, history suggestive of risk to sexually transmitted infections, past history, family history and treatment history were also noted.

A thorough examination of both general and systemic carried out. Dermatological examination including the number, site, morphology and

distribution of primary and secondary lesions then examination of oral and genital mucosa, scalp, hair, nail, palms and soles carried out. Based on this patients were classified as having infective, inflammatory, neoplastic and other diseases.

Investigations like haemogram, blood sugar, blood VDRL, liver function test, urine examination, Mantoux test and CD4 count were done in all cases. Scraping for fungus, appropriate staining and biopsy were done in relevant cases.

All the patients were given symptomatic treatment. Details thus obtained were compiled, tabulated and statistically summarized.

Observations and Results

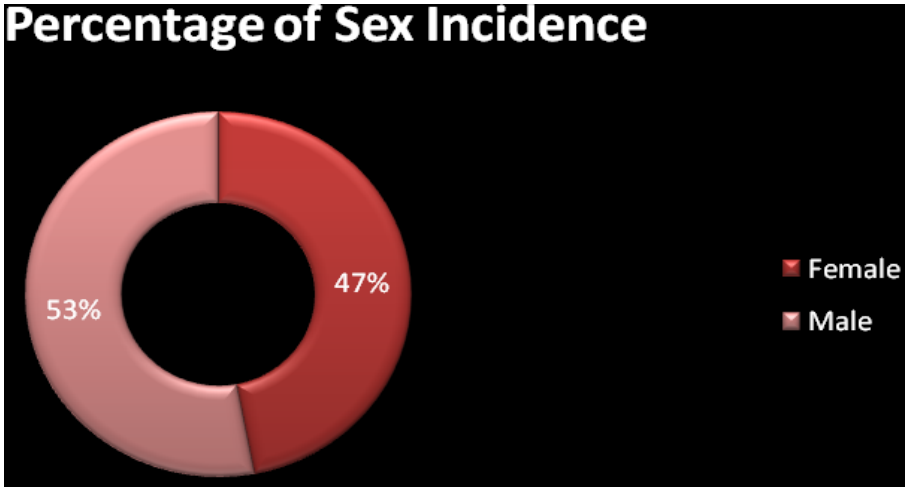
OBSERVATIONS AND RESULTS

After analyzing 100 HIV patients with mucocutaneous manifestations the following observations were made.

Sex incidence

Sex	Frequency	Percent
Female	47	47
Male	53	53
Total	**100**	**100**

Graphical Representation of Sex Incidence:

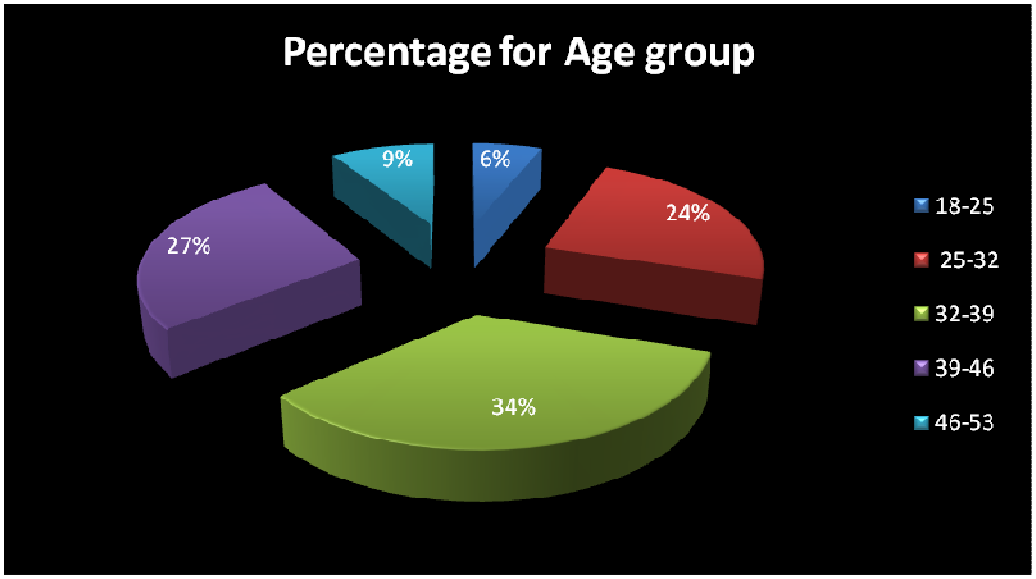


Out of 100 cases studied 57(57%) were males and 47(47%) were females. This indicates a male preponderance.

Age incidence

Age	No. of Persons	Percentage
18-25	6	6
25-32	24	24
32-39	34	34
39-46	27	27
46-53	9	9
Total	**100**	**100**

Graphical representation of Age incidence

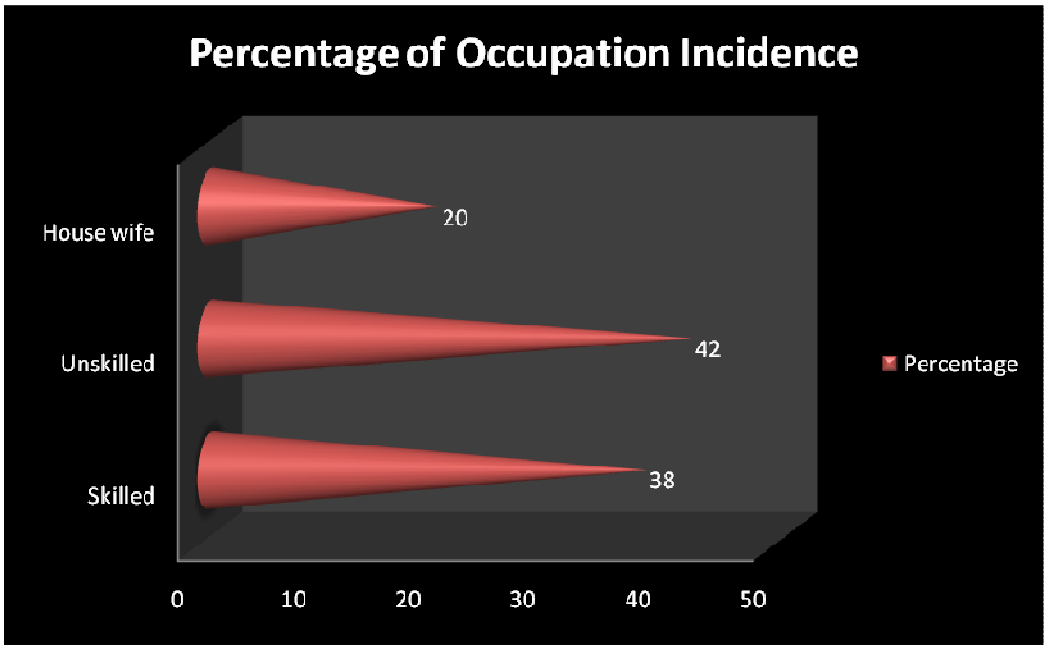


Maximum incidence was noted in the age group between 32-39 years (34%).Minimum incidence was noted in the age group between 18-25 years (6%).

Incidence in Occupation

Occupation	No. of Persons	Percentage
Unskilled	42	42
Skilled	38	38
House wife	20	20
Total	**100**	**100**

Graphical representation of Various Occupation

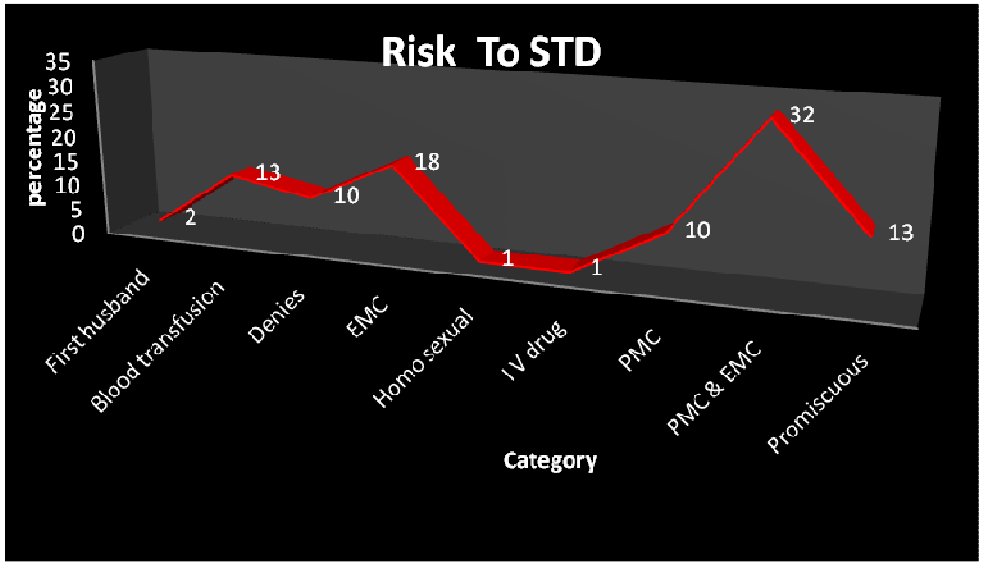


Out of the 100 cases studied, 42 (42%) were unskilled workers, 38(38%) were skilled workers and 20(20%) were house wife. Maximum incidence was observed in unskilled workers.

Risk to STD

Category	Frequency	Percentage
PMC & EMC	32	32
EMC	18	18
Blood transfusion	13	13
Promiscuous	13	13
PMC	10	10
First husband	2	2
Homo sexual	1	1
I V drug	1	1
Denies	10	10
Total	**100**	**100**

Graphical Representation for risk to STD

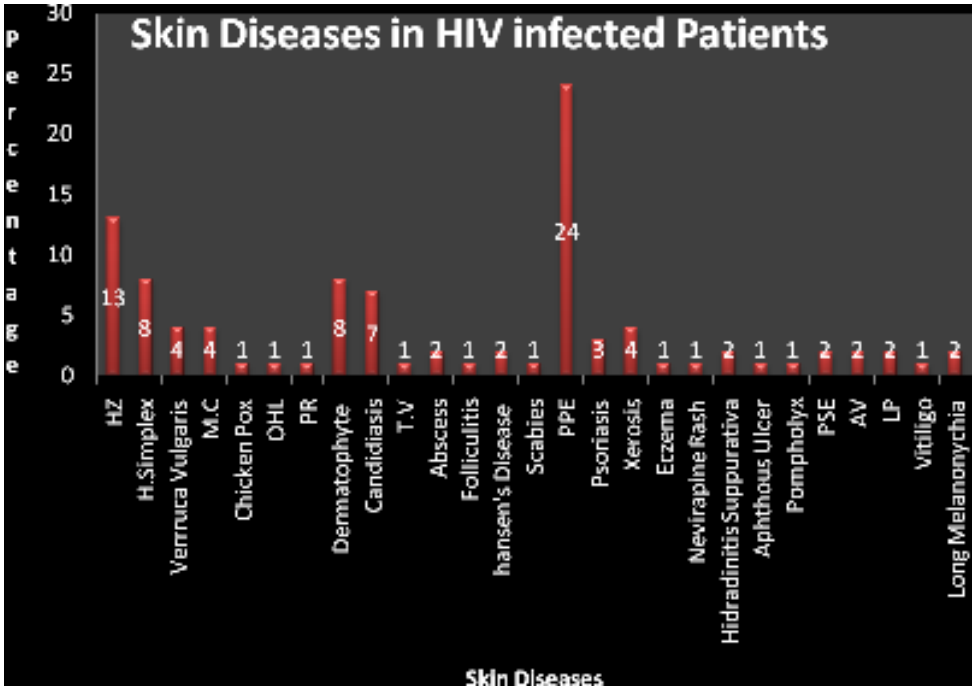


In 100 Patients observed 2% had received HIV from their first husband, 13% received infection through blood transfusion, 10% of them deny the fact that they had extra marital or premarital contact, 18% had extra marital contact, 1% had homosexual contact, 1% had Intravenous drug use, 10% had premarital contact, 32% had premarital contact and extra marital contact and 13% were promiscuous individuals.

Skin Diseases in HIV Infected Patients

Viral Infection		
Skin Disease	**Frequency**	**Percentage**
HZ	13	13
H.Simplex	8	8
Verrruca Vulgaris	4	4
M.C	4	4
Chicken Pox	1	1
OHL	1	1
PR	1	1
Fungal Infection		
Dermatophyte	8	8
Candidiasis	7	7
T.V	1	1
Bacterial Infection		
Abscess	2	2
Folliculitis	1	1
Hansen's Disease	2	2
Parasitic Infection		
Scabies	1	1
Inflammatory Conditions		
PPE	24	24
Psoriasis	3	3
Xerosis	4	4
Eczema	1	1
Nevirapine Rash	1	1
Miscellaneous		
Hidradenitis Suppurativa	2	2
Aphthous Ulcer	1	1
Pompholyx	1	1
PSE	2	2
AV	2	2
LP	2	2
Vitiligo	1	1
Nail Changes		
Long Melanonychia	2	2

Graphical Representation for Skin Disease In HIV Infected Patients



Out of the 100 cases studied Herpes zoster (13%) is the most common infectious disease and pruritic popular eruption (24%) is the most common non-infectious condition.

In 100 cases 13 cases were Herpes zoster (13%). Of which 8 cases were multidermatomal Herpes zoster. One case was associated with psoriasis. Next common viral infection is Herpes simplex, 8 cases (8%).Of which genital Herpes simplex was 5 cases and oral Herpes simplex was 3 cases. Extensive genital herpes was seen in 2 cases. There were 4 cases (4%) of Verruca vulgaris and 4 cases (4%) of Molluscum contagiosum. One case of Molluscum contagiosum was associated with plane wart. One case (1%)

was Oral hairy leukoplakia (OHL). There was one case (1%) of chicken pox. There was also one case (1%) of Pityriasis rosea.

Fungal infections are the next common infectious disease. Out of the 100 cases 8(8%) were Dematophytic infection. There were 5 cases of Tinea corporis, 2 cases of Tinea unguium and one case of Tinea cruris. Candidiasis is the next common fungal infection. There were 7 cases (7%) of candidiasis. There were 4 of cases oral candidiasis, 2 cases of vulvo vaginal candidiasis and one case of oesophageal candidiasis. There was one case (1%) of Tinea versicolor.

Bacterial infections are the 3rd common infectious disease, 5 cases (5%). There were 2 cases of multiple skin abscess and one case of folliculitis. Also there were 2 cases of Hansen's disease, one case was Borderline Tuberculoid with type 1 lepra reaction and the other was Histoid Hansen.

Out of 100 cases there was only one case (1%) of scabies.

The most common skin condition is Pruritic papular eruption (PPE). There were 24 cases (24%) of Pruritic papular eruption. Out of this one case was associated with candidal intertrigo . Pruritic papular eruption was associated with oral candidiasis and seborrheic dermatitis in one case. There was one case associated with Tinea barbae. One was associated with OHL,

one with photosensitive eczema, one with melanonychia, one with melasma and one case with both melasma and melanonychia.

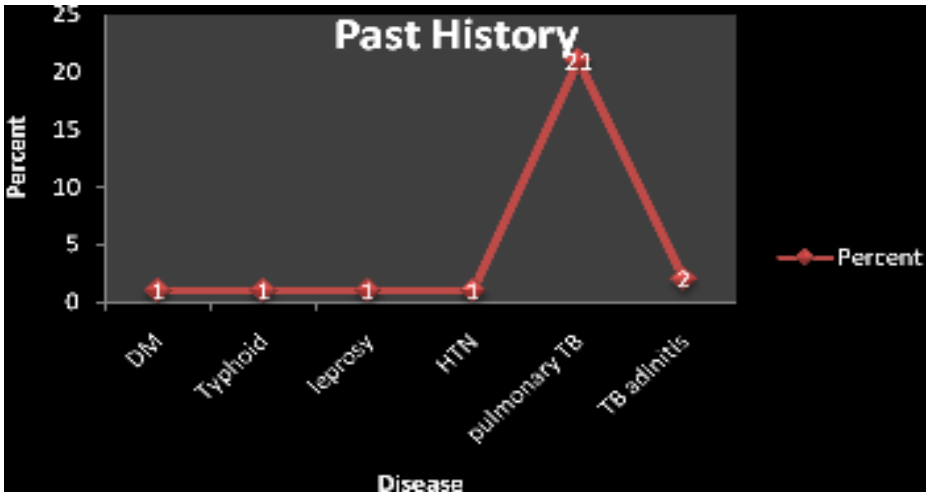
There were 4 cases (4%) of Xerosis. One was associated with asteatotic eczema and other one was with seborrheic dermatitis. There were 3 cases (3%) of Psoriasis. One case was associated with angular chelitis. There were 2 cases of photosensitive eczema and one case of Pompholyx with Melasma. There was one case of Aphthous ulcer, one case of Eczema and one case of Nevirapine rash.

In miscellaneous conditions there were 2 cases (2%) of Hidradenitis suppurativa. Other conditions were 2 cases (2%) of Acne vulgaris, 2 cases of Lichen planus and one case of vitiligo.

PAST HISTORY

Disease	Frequency	Percent
pulmonary TB	21	21
TB adinitis	2	2
DM	1	1
Typhoid	1	1
leprosy	1	1
HTN	1	1
None	73	73
Total	**100**	**100**

Graphical Representation of Past history

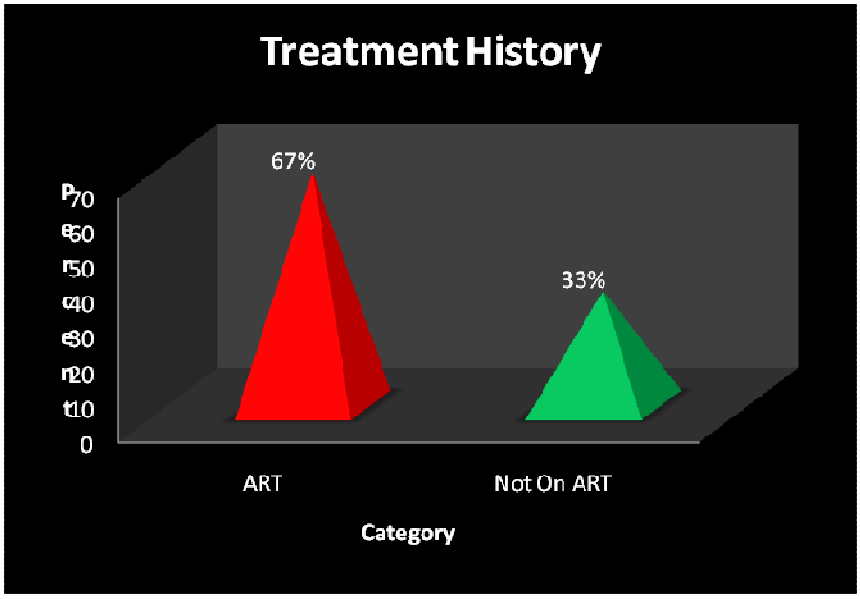


Out of the 100 cases 21(21%) had history of Pulmonary Tuberculosis. Two patients had history of tuberculous cervical lymphadenitis. One has Diabetes mellitus and one has Hypertension. History of Typhoid fever was recorded in one patient and one had history of Leprosy. Out of 100 73 cases (73%) had no history of any significant disease in past.

Treatment History

Category	Frequency	Percent
ART	67	67
Not On ART	33	33
Total	**100**	**100**

Graphical Representation for Treatment History:

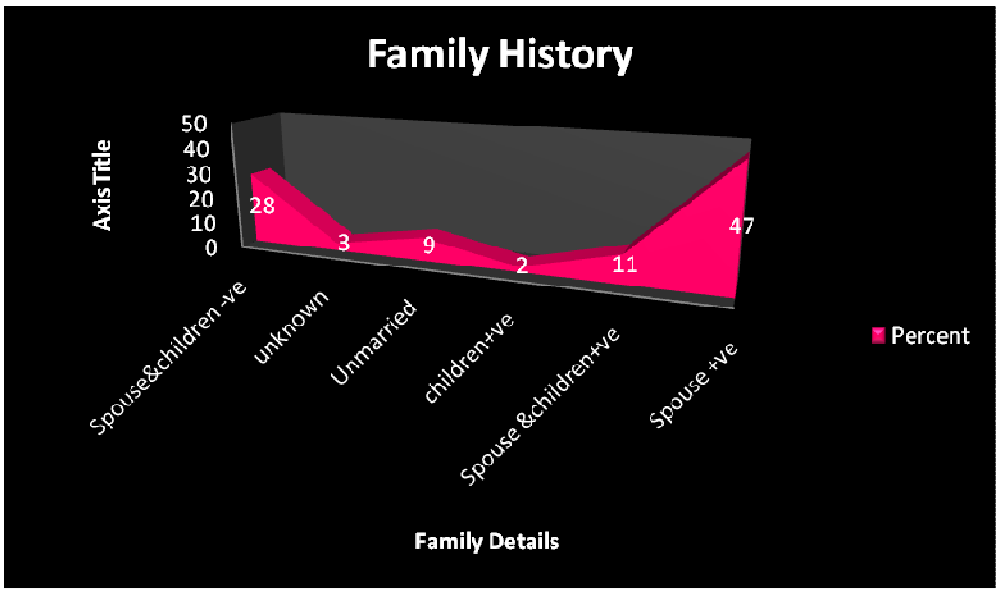


Out of the 100 patients 67(67%) was on ART and 33(33%) was not on ART.

Family history

Family Details	Frequency	Percent
Spouse +ve	47	47
Spouse & children +ve	11	11
Spouse & children -ve	28	28
Children +ve	2	2
Unmarried	9	9
unknown	3	3
Total	**100**	**100**

Graphical Representation for Family History

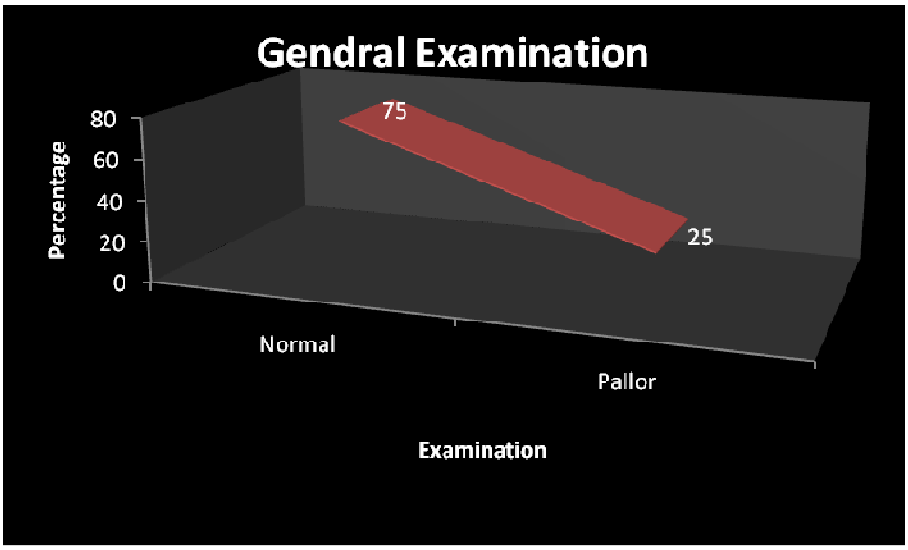


Out of the 100 patients spouse was HIV reactive in 47cases (47%). In 11 cases (11%) both spouse and children were HIV reactive. Only children were positive in 2 cases. Out of this 9 patients (9%) were unmarried. Both spouse and children were HIV nonreactive in 28 cases (28%).The status was unknown in 3 cases (3%).

General Examination

Examination	Frequency	Percent
Normal	75	75
Pallor	25	25
Total	**100**	**100**

Graphical Representation for General Examination



Out of these 100 cases pallor was observed in 25 patients (25%).No abnormality in general examination in 75 patients (75%).

Systemic examination

Systemic examination was normal in all patients.

Investigations

1. Complete haemogram: 25 patients had anaemia.
2. Blood sugar: normal in all patients
3. Blood VDRL: Non Reactive in all patients.
4. Liver function test: One patient had elevated bilirubin, SGOT, SGPT and ALP values.
5. Urine examination: Normal in all patients.
6. MantouxTest: Negative in all patients.
7. CD4+ count was done by flowcytometry in all HIV positives.

Tabular representation of CD4+ count:

CD4+ count	No. of Persons	Percentage
<100	12	12
100-200	27	27
201-350	33	33
351-500	17	17
>500	11	11
Total	**100**	**100**

8. Scraping for fungus: Pseudo hyphae were seen in 7 cases. Hyalinised septate branching hyphae with spores were seen in 11 cases.
9. Staining: Multinucleated giant cells were seen in17 cases. Molluscum bodies were observed in 4 cases. AFB staining for Mycobacterium leprae was positive in 1 case. Gram positive cocci were seen in 4 cases.
10. Biopsy: Biopsy was done in 9 cases. 3cases were psoriasis.2 case were Lichen planus. One case was Histoid Hansen. One case was Borderline tuberculoid Leprosy with type 1 lepra reaction. One case was Verruca vulgaris and one was Verruca plana.

Herpes Zoster

Herpes Simplex

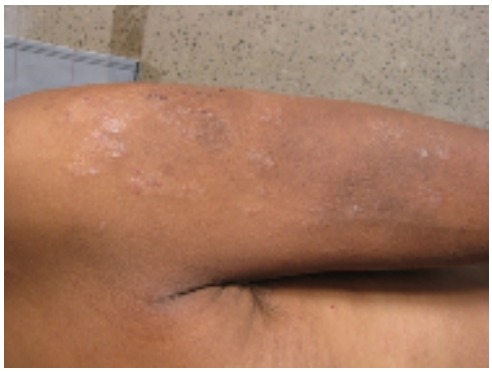
Verruca Vulgaris

Molluscum Contagiosum

OHL

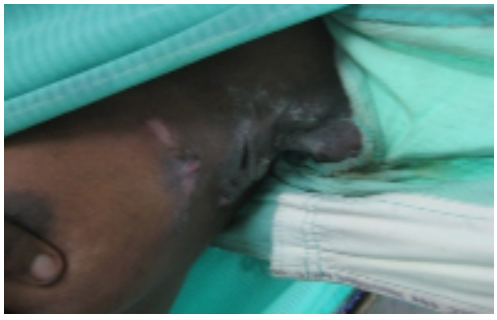
PPE

Tinea Corporis



Tinea Unguium

Hidradenitis Suppurativa



Candidiasis

Psoriasis

Abscess

Hansen's disease (Type 1 Reaction)

Xerosis

PSE

Discussion

DISCUSSION

In our study maximum incidence of cutaneous manifestation was noted in the age group between 32- 39(34%). This is similar to that reported in literature.[277] From our analysis we noted a male predominance in HIV population. Jindal N, et al. quoted a female predominance in cutaneous manifestations.[277] We observed a higher ratio of HIV infection in unskilled workers. This may be due to Coimbatore being an industrial area were migrant population is high. It was observed that most of the seropositive men had acquired the infection from commercial sex workers and had passed it on to their spouse through unprotected sexual intercourse. 91% infected person were married in this observation. Heterosexual transmission(75%) was the highest mode of transmission in our analysis, similar to other studies.[277,278] We also noted that HIV transmission through infected blood transfusion(13%) was somewhat low in our population compared to other study.[279] A good number of the patients (67%) were registered for Anti retroviral Therapy in our case study.

Because of immunosuppression, HIV positive patients have various cutaneous manifestations expected than others. In this analysis multiple lesions were observed in 16% of patients, this is to some extent less compared to other study.[277] In most of our patients the etiology was non infectious and the most common lesion was Pruritic papular eruption (24%). This is in concurrence with the findings of Ajay S et al .[280] Next to Pruritic

papular eruption the maximum etiology was viral infection mainly Herpes zoster (13%) of which 8 patients had multi dermatomal Herpes zoster similar to other study.[277] Its is important to recognize the wide and varied spectrum of Herpes zoster in HIV patients. In our analysis we had 8% of the patients with Herpes Simplex and 8% had Dermatophyte infection, our observation was similar to that of Kumarasamy et al[276] and Spira et al.[280] As most of the patient on ART the mucocutaneous Candidiasis was observed in 7% compared to Singh. H et al[281] study. Extensive Molluscum contagiosum was the manifestation of 4% of patients. This is low compared to the observations of Jindal. N.et al (13%). [277] We observed that 4% of patients had common wart, Compared to other studies[276,277] there was no case of Genital wart in this analysis.

In our analysis 4% patients had Xerosis, this may be due to the disease itself or due to chronic malnutrition. India is a developing country and malnutrition is a common problem, this value is somewhat less compared to other Indian studies.[271, 278] We also found out that 3% of patients had Psoriasis.

Although the prevalence of leprosy is falling in India, its recognition, especially tuberculoid leprosy, and diagnosis is important in the management of Immune Reconstitution Inflammatory syndrome (IRIS). [282] The disease was not so uncommon in our place. We detected 2 cases of

Hansen's disease. One case was presented as Immune Reconstitution Inflammatory syndrome (IRIS), after one month of ART. Previously undetected leprosy has been diagnosed after onset of IRIS in HIV patients. [283, 284] It was a case of Borderline Tuberculoid Leprosy with type 1 lepra reaction. The other case was Histoid Hansen.

We observed 2 cases of Hidradenitis suppurativa which is less compared to Nair SP et al study.[285] Although most of our study patients were on ART only 2 patients had longitudinal melanonychia. Sharma A et al reported 14% of patients with nail pigmentation.[286] Other condition observed in the present study were Oral hairy leukoplakia (1%) which is not as common as reported by Nair SP et al[285] may due to the fact that most of the patients were on ART. There were 2 cases of skin abscess and one case of folliculitis, this was less compared to study by Bhandary P G, et al.[287] There was one case of Nevirapine rash ,this was low compared to other study.[286] There was one case of scabies. Jindal N, et al. was observed 4 patients (10%) with scabies.[277] There were 2 cases of Lichen planus, this was correlating with other study.[277] There were 2 cases of Seborrheic dermatitis, this was less compared to an earlier study.[287] Other conditions observed in our study were Aphthous ulcer, Pompholyx, Photosensitive eczema, Pityriasis rosea, Acne vulgaris, Chicken pox, Eczema, and Vitiligo. No case of cutaneous Neoplasia was observed.

Majority of skin disease was observed when the CD4+ count falls below 350. There were 33 patients with CD4+ count 201-350 and 27 patients with CD4+ 100-200. In patients with Pruritic Papular Eruption the CD4+ count was in the range of 68-280. In case of Herpes zoster the range of the CD4+ count was 46-493. Dermatophyte infection was seen in the range of 89-658. In Candidiasis the CD4+ count was low in most of the patients in the range of 80-335.In most of the patients with Herpes simplex there was low CD4+ count. Oral hairy leukoplakia was seen in the range of 64-180. This was comparable with the study by Shobhana A.et al.[8] There were 25% of patients with anemia, similar to other study.[286]

Conclusion

CONCLUSION

HIV/AIDS involves almost all the organs in the body. Mostly Infections and Malignancies are seen. Many a time the evolution of the cutaneous manifestation gives a clue to the diagnosis of HIV and stage of the disease. At times depending upon the CD4+ count, there is specific dermatological manifestations. Mostly cutaneous manifestations are clear markers of HIV disease, and many a time they may be the first lesion to be identified in an otherwise normal HIV reactive patient.

In the present study we have attempted to identify the common cutaneous markers of HIV/AIDS and we have found out that indeed there is a correlation between certain dermatological conditions and HIV spectrum. Various previous studies and our study are correlating each other. Most common presentation in our study is Pruritic Papular Eruption(24%).Then Herpes Zoster(13%), Herpes simplex(8%), Dermatophytosis(8%) and Candidiasis(7%) was observed. Incidence of Candidiasis was probably low because patients were coming in the earlier stage or had taken symptomatic treatment from outside.

To conclude cutaneous manifestations are the most common presentation of HIV disease and Pruritic Papular Eruption, and Herpes Zoster are the tell tale evidences of HIV disease.

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PROFORMA

OP No: DATE:

Name :

Age :

Sex :

Address :

Occupation :

Income:

Complaints : Duration:

1.

2.

3.

History of Sexual exposure /Risk to STD

Present History:

Evolution and spread of lesion
H/o recurrence
H/o Mucosal involvement
H/o prior drug intake
H/o fever/ weight loss/ diarrhoea
H/o altered taste/ dysphagia

Past History:

Family history:

Similar illness in family

Treatment history:

Personal history:

General examination:

Systemic examination:

Dermatological examination:

Skin:

Type of lesion

Distribution

Mucosa:

Oral

Genital

Scalp:

Hair and nail:

Palms and Soles:

SKIN DISEASES			
Infective	Bacterial	Bacterial folliculitis	
		Boil	
		Impetigo	
		Skin abscess	
		Syphilis(1º & 2º)	
		Tuberculosis	
		Atypical mycobacterium	
		Bacillary angiomatosis	
	Fungal	Dermatophytosis	
		Pityriasis versicolor	
		Candidiasis	
		Cryptococcosis	
		Penicilliosis	
	Viral	Herpes Zoster	
		Herpes Simplex	
		Molluscum Contagiosum	
		Wart	
	Arthropod	Scabies	
Inflammatory		Cutaneous drug eruption	
		Eosinophilic folliculitis	
		Seborrhoeic dermatitis	
		Psoriasis	
		Atopic eczema	
		Xerosis,icthyosis and asteatotic dermatitis	
		Pruritic papular eruption	
\Neoplastic		Kaposi's sarcoma	
		Non-Hodgkin's Lymphoma	
Miscellaneous		Lipodystrophy syndrome	
		Conditions involving nails and hairs	

INVESTIGATION

Blood:

Hb

TC

DC

ESR

Platelet count

Blood VDRL :

LFT :

Billirubin... Total:
Direct:
SGOT :
SGPT :
ALP :
Sr Albumin :
Globulin :

Urine Examination :

Mantoux Test :

CD4 Count :

Scraping for Fungus :

Staining :

Biopsy :

Culture :

Follow up:

ABBREVIATIONS

AIDS	Acquired immunodeficiency syndrome
HIV	Human immunodeficiency virus
KS	Kaposi's sarcoma
ART	Anti retroviral therapy
IDU	Intra venous drug use
STD	Sexually transmitted disease
ARV	Antiretroviral drugs
MTCT	Mother to child transmission
RT PCR	Reverse transcriptase polymerase chain reaction
CD	Cluster differentiation
OHL	Oral hairy leukoplakia
MAC	Mycobacterium avium complex
CMV	Cytomegalovirus
PCP	Pneumocystis carinii pneumonia
OPC	Oropharyngeal candidiasis
HSV	Herpes simplex virus
EBV	Epstein barr virus
VZV	Varicella zoster virus
HPV	Human papilloma virus

HBV	Hepatitis B virus
HCV	Hepatitis C virus
TB	Tuberculosis
BA	Bacillary angiomatosis
OI	Opportunistic infection
HS	Hydradenitis suppurativa
MC	Molluscum contagiosum
PR	Pityriasis rosea
HZ	Herpes zoster
TV	Tinea versicolor
PPE	Pruritic papular eruption
PSE	Photo sensitive eczema
AV	Acne vulgaris
LP	Lichen planus
DM	Diabetes mellitus
HTN	Hypertension
IRIS	Immune reconstitution inflammatory syndrome

Master Chart

SI NO	AGE	SEX	LOCATION	OCCUPATION	RISK TO STD	PAST HISTORY	FAMILY HISTORY		ART	DERMATOLOGICAL EXAMINATION									SKIN DISEASES	CD4 COUNT	BIOPSY
							SPOUSE	CHILDERN		SKIN		MUCOSA			HAIR	NAIL	PALMS	SOLES			
										TYPE OF LESION	DISTRIBUTION	ORAL	GENTIAL	SCALP							
1	40	M	CBE	UNSK	EMC	NIL	NR	NR	ART	Vesicles	Both LL	Glossitis	N	Silvery White Scale	N	N	N	N	H Z, Psoriasis	234	-
2	31	F	CBE	UNSK	PMC	NIL	R	NR	ART	Papules with central umbilication	Right face , eye lid,	N	N	N	N	N	N	N	MC, plane wart	178	V.plana
										Flat topped papules	Fore head, neck & back										
3	37	M	CBE	SK	EMC	NIL	R	NR	No	N	-	White hairy plaque on the right lateral side of tongue	N	N	N	N	N	N	OHL	150	-
4	42	M	CBE	SK	EMC	NIL	NR	NR	No	N	-	Glossitis	N	N	N	N	N	N	Oesophageal Candidiasis	90	-
5	31	M	CBE	SK	MSM	NIL	R	NR	ART	papules central umbiliction	Face	N	N	N	N	N	N	N	M C	64	-
6	33	M	CBE	UNSK	PMC & EMC	NIL	NR	NR	No	shiny nodules stuck on appearance	Face, Ear,	N	N	N	N	N	N	N	Histoid hansen	370	histoid
7	42	M	POL	SK	PM	NIL	R	NR	No	Groups of vesicles clear fluid	left UL	N	N	N	N	N	N	N	H Z	270	-
8	45	M	CBE	SK	PMC	NIL	R	R	ART	N		N	N	N	N	HPG	N	N	Onychomycosis	112	-
9	30	F	CBE	SK	EMC	NIL	NR	NR	No	Groups of vesicles with clear fluid	Right thigh	N	N	N	N	N	N	N	H Z	290	-
10	55	M	TIR	UNSK	Denies	NIL	R	NR	ART			Multiple ulcer with erythematous base on buccal muocsa	N	N	N	N	N	N	Aphthous ulcer	200	-
11	45	M	CBE	UNSK	EMC&PMC	NIL	NR	NR	ART	Wheals	Back	N	N	N	N	LM	N	N	Urticaria,LM	211	-
12	29	F	TIR	HW	EMC	NIL	NR	R	No	papules with Excoriation	Face, UL, back & legs	N	N	N	N	N	N	N	PPE	180	
13	33	M	CBE	SK	EMC&PMC	NIL	R		No	central clearing & raised borders	multiple papules	N	N	N	N	N	N	N	T. barbae,PPE	134	-
14	34	F	CBE	SK	Denies	NIL	R	NR	ART	papules with Excoriation	fore arm & legs	N	N	N	N	N	N	N	PPE	220	
15	29	F	TIR	SK	PMC	NIL	R	R	ART	central clearing &raised borders	left of abdomen	N	N	N	N	N	N	N	T. corporis	421	-
16	40	F	ANR	UNSK	EMC&PMC	NIL	R	NR	ART	papules with Excoriation	fore arms	N	N	N	N	N	N	N	PPE	180	
17	37	F	CBE	SK	EMC	NIL	R	No	ART	Groups of vesicles with clear fluid	left UL & upper back	N	N	N	N	N	N	N	HZ	133	-
18	34	F	SLM	HW	EMC&PMC	PT	R	NR	ART	Multiple pustules& sinuses	axillae & gluteal region	N	N	N	N	N	N	N	Hidradenitis suppurativa	406	-
19	37	M	CBE	SK	EMC&PMC	NIL	R	R	No	erythematous plaques silvery scales	elbows ,scalp & knees	N	N	N	N	N	N	N	psoriasis	421	Psoriasis
20	30	F	CBE	HW	PMC	PT	NR	NR	ART	vesicles , hyper pigmented patch	all fingers,cheeks	N	N	N	N	N	N	N	pompholyx,melasma	494	-
21	35	F	POL	HW	BT	NIL	NR	NR	No	Multiple papules	fore arms & legs	White linear plaque over lateral part of tongue	N	N	N	N	N	N	OHL,PPE	180	-
22	26	M	CBE	UNSK	BT	NIL	NR	NR	No	Groups of vesicles with clear fluid	left thigh & back	N	N	N	N	N	N	N	HZ	260	-
23	40	M	TIR	UNSK	Denies	NIL	R	NR	ART	Multiple papules & erosions	Legs	N	N	N	N	N	N	N	PPE	127	
24	28	F	CBE	UNSK	BT	PT	R	R	ART	Dry scaly hypo pigmented patches	forearms & legs	N	N	N	N	N	N	N	Xerosis,Asteatotic eczema	122	-
25	52	M	CBE	UNSK	EMC&PMC	TYPH	R	NR	ART	Multiple papules & erosions	forearms & legs	N	N	N	N	N	N	N	PPE	210	-
26	50	M	CBE	UNSK	PM	NIL	R	NR	No	multiple papules	Face	N	N	N	N	N	N	N	PPE	130	-
27	34	M	ERD	SK	EMC	HTN	R	NR	ART	Scaly plaque	right wrist	N	N	Fine greasy yellowish scale	N	N	N	N	Seborrheic dermatitis ,Xerosis	408	-
28	47	F	CBE	HW	BT	TB_LN,D	NR	NR	ART	Multiple papules & erosions	fore arms & legs	N	N	N	N	N	N	N	PPE	160	-
29	37	F	CBE	SK	PMC	NIL	R	NR	ART			N	Multiple Shallow ulcers with polycyclic boarders	N	N	N	N	N	Herpes Simplex	474	-
30	39	M	CBE	SK	PMC	NIL	R	R	ART	Multiple pustules& sinuses	Axillae	N	N	N	N	N	N	N	Hidradenitis suppurativa	282	-
31	67	M	TIR	SK	EMC&PMC	NIL	NR	NR	ART	scaly plaque	forearms & legs	Curdy white plaque on tongue	N	N	N	N	N	N	Oral Candidiasis,Ichthyosis	80	-
32	30	M	SLM	UNSK	PM	NIL	UNM	-	ART	Warty papules	penis & scrotum	N	N	N	N	N	N	N	Common Wart	544	-
33	40	F	CBE	HW	EMC	PT	NR	NR	No			white plaque over tongue	N	N	N	N	N	N	Oral Candidiasis	234	-
34	29	F	TIR	HW	EMC&PMC	NIL	R	NR	ART	Abscess with discharge	left lower back	N	N	N	N	N	N	N	Skin abscess	123	-
35	34	F	CBE	SK	PM	NIL	R	NR	No	Multiple papules	Face & neck	N	N	N	N	N	N	N	PPE	126	
36	58	M	CBE	UNSK	EMC&PMC	NIL	NR	NR	No	Groups of vesicles with clear fluid	left chest & back	N	N	N	N	N	N	N	HZ	184	-
37	38	F	KUR	SK	Denies	NIL	R	R	ART	hyper pigmented patch	Fore head & back	N	N	N	N	N	N	N	PSE	444	-
38	40	M	CBE	SK	BT	NIL	R	R	ART	multiple pustules	scrotum	N	N	N	N	N	N	N	Skin abscess	216	-
39	38	F	CBE	HW	EMC	NIL	R	R	ART	Multiple papules	forearms & legs	N	N	N	N	N	N	N	PPE	96	-
40	32	F	TIR	HW	EMC&PMC	NIL	R	NR	ART	hypopigmented macules & patches	neck & back	N	N	N	N	N	N	N	TV	260	-
41	33	M	TIR	SK	PM	NIL	R	NR	No	multiple pustules	gluteal & perianal region	N	N	N	N	N	N	N	folliculitis	308	-
42	24	F	CBE	HW	1st Husband	NIL	NR	NR	No	plaques with collarette of scales	forearms,back & legs	N	N	N	N	N	N	N	PR	618	-
43	36	F	CBE	UNSK	Denies	NIL	R	NR	ART	Groups of vesicles with clear fluid	Right UL	N	N	N	N	N	N	N	HZ	97	-
44	35	M	POL	SK	PM	PT	UNM	-	ART	Groups of vesicles with clear fluid	Left mandibular & neck	N	N	N	N	N	N	N	HZ	421	-
45	42	M	CBE	SK	EMC&PMC	NIL	R	NR	ART	Multiple papules	Fore arm	N	N	N	N	N	N	N	PPE	130	-
46	47	F	CBE	UNSK	Denies	NIL	R	-	ART	Multiple papules	forearms & legs	N	N	N	N	N	N	N	PPE	196	-
47	45	F	CBE	UNSK	BT	NIL	NR	NR	ART	N		N	Curdy white discharge	N	N	N	N	N	VVC	283	-
48	30	F	TIR	HW	1st Husband	NIL	NR	R	ART	Multiple papules	forearms, legs & back	N	N	N	N	N	N	N	PPE	157	-
49	54	M	CBE	SK	EMC&PMC	NIL	NR	NR	ART	Multiple erythematous Papules	All over the body	N	N	N	N	N	N	N	Nevirapine rash	213	-
50	30	F	SLM	HW	BT	NIL	NR	NR	ART	hyper pigmented patch	both cheeks	N	Curdy white discharge	N	N	N	N	N	Melasma, VVC	394	-
51	37	M	POL	SK	PM	PT	R	NR	ART	Lichenified Scaly plaque	left leg	N	N	N	N	LM	N	N	Eczema, LM	507	-

52	40	F	TIR	HW	EMC	NIL	NR	NR	ART	Dry scaly patches	forearms, chest & abdomen	N	N	N	N	N	N	N	xerosis	188	-
53	29	F	TIR	UNSK	EMC&PMC	PT	R	NR	ART	papules, Pustules & comedones	Face	N	N	N	N	N	N	N	AV	439	-
54	32	M	CBE	UNSK	PM	NIL	NR	NR	ART	Multiple papules	forearms, legs & face	N	N	N	N	N	N	N	PPE	156	-
55	42	M	PKD	UNSK	EMC&PMC	NIL	R	NR	ART	central clearing & raised borders	Left thigh & buttocks	N	N	N	N	HPG	N	N	T. corporis, Onychomycosis	427	-
56	25	F	TIR	SK	BT	NIL	NR	NR	ART	papules & hypopigmented patches	forearms, legs & back	N	N	N	N	N	N	N	PPE & PSE	225	-
57	42	M	SLM	SK	PM	PT	-	-	ART	Multiple erythematous Papules	Chest, back, abdomen & UL	N	N	N	N	N	N	N	Chicken pox	512	-
58	41	M	CBE	SK	PM	NIL	-	-	ART	hyper pigmented Scaly plaque	dorsum of right hand	N	N	N	N	N	N	N	Eczema	469	-
59	36	M	CBE	SK	EMC	NIL	NR	NR	No	erythematous plaques , silvery scale	scrotum	Angular Chelitis	N	N	N	N	N	N	Psoriasis, Angular Chelitis	386	Psoriasis
60	32	F	CBE	SK	Denies	NIL	UNK	NR	ART	central clearing &raised borders	axillae & groins	N	N	N	N	N	N	N	T. corporis & T. cruris	664	-
61	32	M	CBE	SK	EMC	PT	NR	NR	No	papules, Pustules & comedones	face & back	N	N	N	N	N	N	N	AV	230	-
62	38	M	SLM	UNSK	EMC&PMC	PT	NR	NR	ART	erosions	lower lip	erosions over buccal mucosa	N	N	N	N	N	N	Herpes Simplex	216	-
63	30	M	CBE	SK	PM	PT	UNM	-	No	multiple verrucous papules	right thumb	N	N	N	N	N	N	N	Common Wart	589	Wart
64	33	M	POL	UNSK	EMC&PMC	PT	NR	NR	ART	central clearing &raised borders	L forearm, face & right leg	N	N	N	N	N	N	N	Common Wart	249	-
65	38	F	POL	UNSK	Denies	NIL	R	NR	ART	violaceous papules & plaques	UL,LL,chest & back	erosions over buccal mucosa	N	N	N	N	N	N	LP	320	LP
66	36	M	CBE	SK	EMC&PMC	NIL	UNM	-	ART	erosions & crusting	lower lip	N	N	N	N	N	N	N	Herpes Simplex	673	-
67	42	F	CBE	UNSK	EMC	NIL	R	NR	No	vesicles with erosions & crusting	right leg & sacral area	N	N	N	N	N	N	N	HZ	210	-
68	31	F	CBE	HW	EMC&PMC	NIL	R	NR	No	multiple verrucous papules	dorsum of both hands	N	N	N	N	N	N	N	Common Wart	207	-
69	31	F	CNR	UNSK	EMC&PMC	NIL	R	NR	ART	multiple papules	face & both forearms	N	N	N	N	LM	N	N	PPE,melanonychia	203	-
70	35	F	TIR	UNSK	Denies	NIL	R	NR	ART	hyperpigmented patch	both UL & face,both cheeks	N	N	N	N	N	N	N	PPE,melasma	243	-
71	45	F	POL	UNSK	EMC&PMC	DM	R	NR	ART	excoriations & hyperpigmentation	bothUL,LL,chest&back	N	N	N	N	N	N	N	scabies	426	-
72	42	M	CBE	UNSK	PMC	NIL	R	NR	ART	multiple papules	face & both forearms	N	N	N	N	N	N	N	PPE	180	-
73	40	F	CBE	UNSK	Denies	TB LN	NR	NR	ART	multiple papules with umbilication	lips &face	N	N	N	N	N	N	N	MC	271	-
74	35	M	SLM	UNSK	EMC	NIL	R	NR	No	N			erosions over glans penis	N	N	N	N	N	Herpes Simplex	210	-
75	43	M	CBE	SK	PMC	HD	R	NR	ART	Hyper pigmented Scaly patch	both lower legs	N	N	N	N	N	N	N	Xerosis	337	-
76	37	M	TIR	UNSK	EMC	PT	R	NR	ART	multiple papules	both fore arms, legs & foot	N	N	N	N	N	N	ITR	N	PPE, Candidiasis	190	-
77	30	M	CBE	SK	PMC	NIL	R	R	ART	erythematous plaque, thickened N	Right forearm, above right eye , back & scalp	N	N	N	N	N	N	N	Bor Tub & reaction type 1	215	Bor Tub,reaction
78	43	M	TIR	UNSK	EMC&PMC	NIL	UNM		ART	Annular scaly patch mutiple papules	Anterior neck face	white plaque over tongue	N	N	N	N	N	N	Seborrheic dermatitis, PPE, oral candidiasis	200	-
79	28	M	TIR	SK	BT	PT	NR	NR	ART	Mutiple hyper pigmented papules	left forehead	N	N	N	N	N	N	N	LP	680	LP
80	50	M	POL	UNSK	BT	NIL	UNM		No	depigmented macules & patches	UL & LL, lips, h&s & scrotum	Glossitis	N	N	N	N	N	DPIG	N	Vitiligo	90	-
81	33	F	CBE	UNSK	EMC&PMC	NIL	R	NR	No	vesicles & pustules with crusting	left side of the face	N	N	N	N	N	N	N	HZ	240	-
82	34	M	POL	SK	BT	PT	R	NR	No	N		white plaque over tongue	N	N	N	N	N	N	Oral Candidiasis	220	-
83	35	F	POL	HW	EMC&PMC	NIL	R	NR	No	Mutiple vesicles & erosions	Left angle of month	N	N	N	N	N	N	N	Herpes Simplex	113	-
84	34	F	CBE	HW	BT	NIL	R	NR	ART	papules, hyperpigmented patches	Fore arm , legs & back, cheeks & nose	N	N	N	N	LM	N	N	PPE, melasma, melanonychia	76	
85	43	F	TIR	HW	EMC&PMC	NIL	R	NR	No	N		N	Multiple Shallow ulcers with polycyclic borders	N	N	N	N	N	Herpes Simplex	220	-
86	36	M	TIR	UNSK	EMC	PT	NR	NR	ART	central clearing &raised borders	gluteal region	N	N	N	N	N	N	N	T. corporis	472	-
87	34	M	TIR	UNSK	EMC	PT	NR	NR	ART	N		white plaque over tongue	N	N	N	N	N	N	Oral Candidiasis	68	-
88	28	F	TIR	HW	EMC&PMC	PT	R	NR	ART	N		N	Multiple Shallow ulcers with polycyclic	N	N	N	N	N	Herpes Simplex	94	-
89	37	F	ERD	UNSK	PM	NIL	UNM	-	ART	grouped vesicles & pustules	right side of chest & axilla	Glossitis	N	N	N	N	N	N	HZ	378	-
90	45	M	TIR	UNSK	PMC	NIL	NR	NR	ART	grouped vesicles & pustules	left thigh medial aspect & leg	Glossitis	N	N	N	N	N	N	HZ	46	-
91	31	F	CBE	UNSK	EMC&PMC	NIL	R	NR	No	grouped vesicles & pustules	left thigh	Glossites	N	N	N	N	N	N	HZ	207	-
92	30	F	CBE	UNSK	PM	NIL	R	R	No	multiple papules	boyh fore arms, back, legs & foot	N	N	N	N	N	N	N	PPE	249	
93	49	M	POL	UNSK	EMC	PT	NR	NR	ART	central clearing &raised borders	back	N	N	N	N	N	N	N	T. corporis	302	-
94	70	M	TIR	UNSK	EMC&PMC	NIL	R	NR	No	central clearing &raised borders	both groin	N	N	N	N	N	N	N	T. cruris	658	-
95	25	F	ERD	UNSK	BT	NIL	R	NR	No	erythematous plaques, silvery scale	Scalp, back of the ear, neck & back	N	N	N	N	N	N	N	Psoriasis	628	Psoriasis
96	39	M	CBE	SK	PM	NIL	UNM	-	ART	central clearing &raised borders	both UL, legs & back	N	N	N	N	N	N	N	T. corporis	89	-
97	32	M	CBE	SK	EMC&PMC	NIL	R	NR	No	multiple hypopigmented patches	both forearms,back &anterior neck	N	N	N	N	N	N	N	PSE	547	-
98	40	F	CBE	HW	EMC&PMC	NIL	R	NR	ART	N			Multiple Shallow ulcers with polycyclic borders	N	N	N	N	N	Herpes Simplex	169	-
99	35	M	CBE	SK	I V drug	PT	UNM	-	ART	multiple papules	both forearms &legs	N	N	N	N	N	N	N	PPE	140	
100	40	F	CBE	HW	EMC&PMC	PT	R	NR	No	multiple papules with umbilication	right eyelid,left cheek,nose &right shoulder	N	N	N	N	N	N	N	MC	68	-

KEY TO THE MASTER CHART

UNSK	Unskilled workers
SK	Skilled workers
HW	House wife
EMC	Extra marital contact
MSM	Male having sex with male
PTB	Pulmonary Tuberculosis
R	HIV reactive
NR	HIV nonreactive
UNM	Unmarried
Unkn	Unknown HIV status
P+	Pallor present
UL	Upper limb
LL	Lower limb
T.barbae	Tinea barbae
T. corporis	Tinea corporis
M	Male
F	Female
CBE	Coimbatore
POL	Pollachi
TIR	Thirpur
ANR	Annoor
SLM	Salem

KUR	Karur
ERD	Erode
PKD	Palakad
CNR	Coonoor
PM	Promiscuous
BT	Blood Transfusion
TYPH	Typhoid
TBLN	Tuberculous Lymphadenitis
HD	Hansen’s Disease
N	Normal
VVC	Vulvo Vaginal Candidiasis
LM	Longitudinal Melanonychia
HPG	Hyper pigmentation
ITR	Intertrigo
DPIG	Depigmentation
Bor Tub	Borderline Tuberculoid